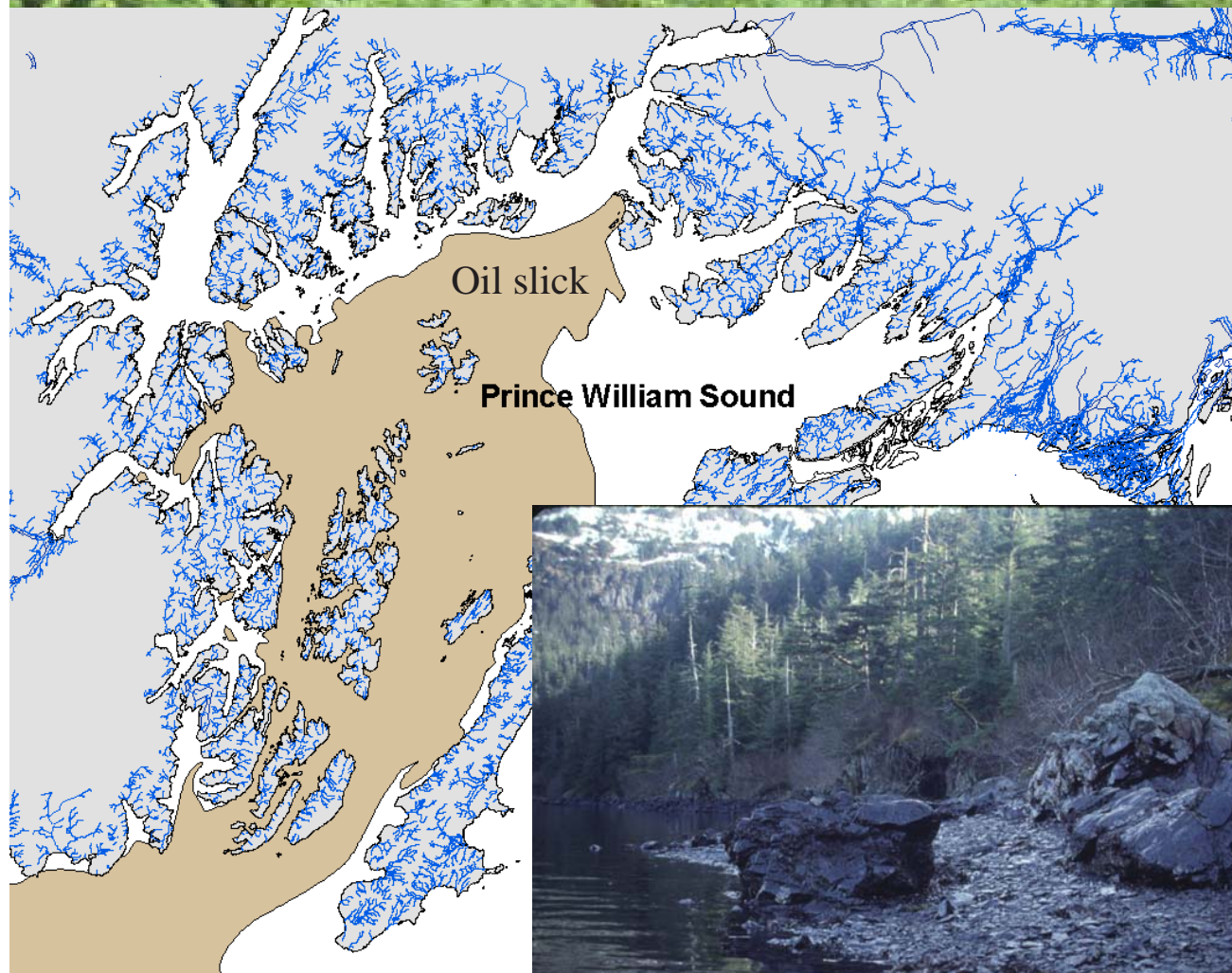


# Pink Salmon Habitat is Recovering a Decade After the *Exxon Valdez* Oil Spill

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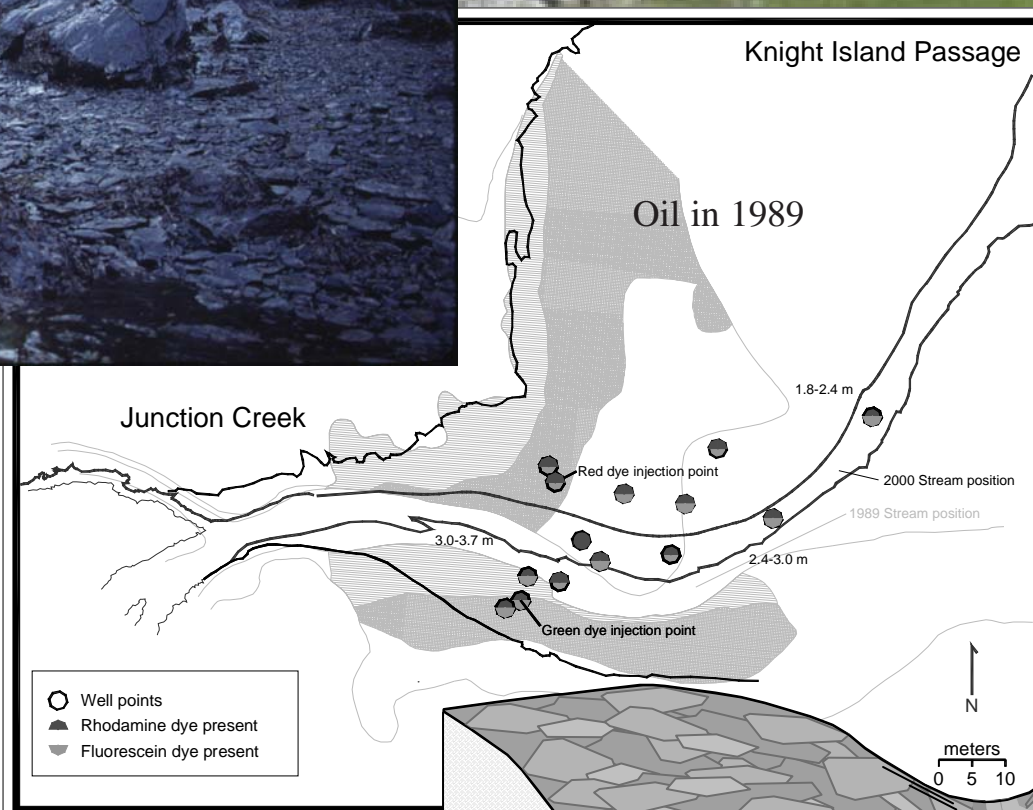
(In press; Trans. Am. Fish. Soc.)



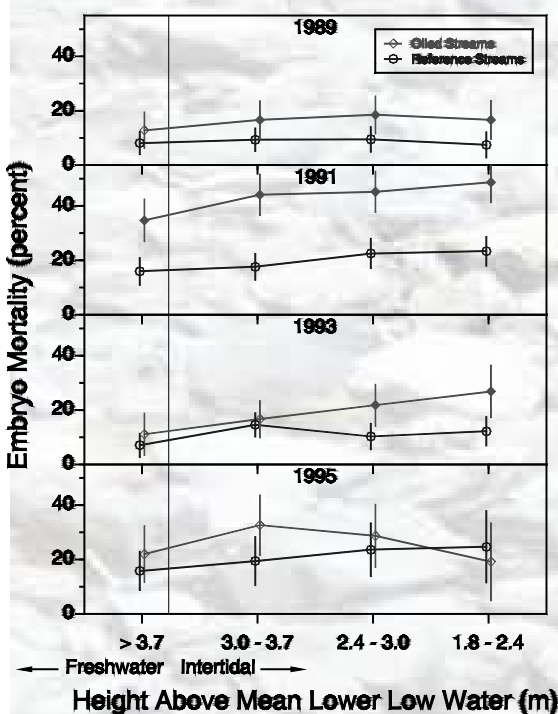
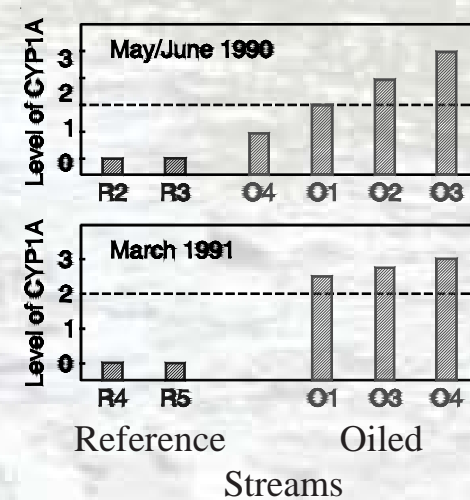
**(1) The problem.** The *Exxon Valdez* oil spill occurred more than a decade ago in Prince William Sound, Alaska.



**(2)** Consequently, many intertidal beaches were coated by oil, including those surrounding streams utilized as spawning habitat by pink salmon (*Oncorhynchus gorbuscha*) (Geiger et al. 1996; Murphy et al. 1999).



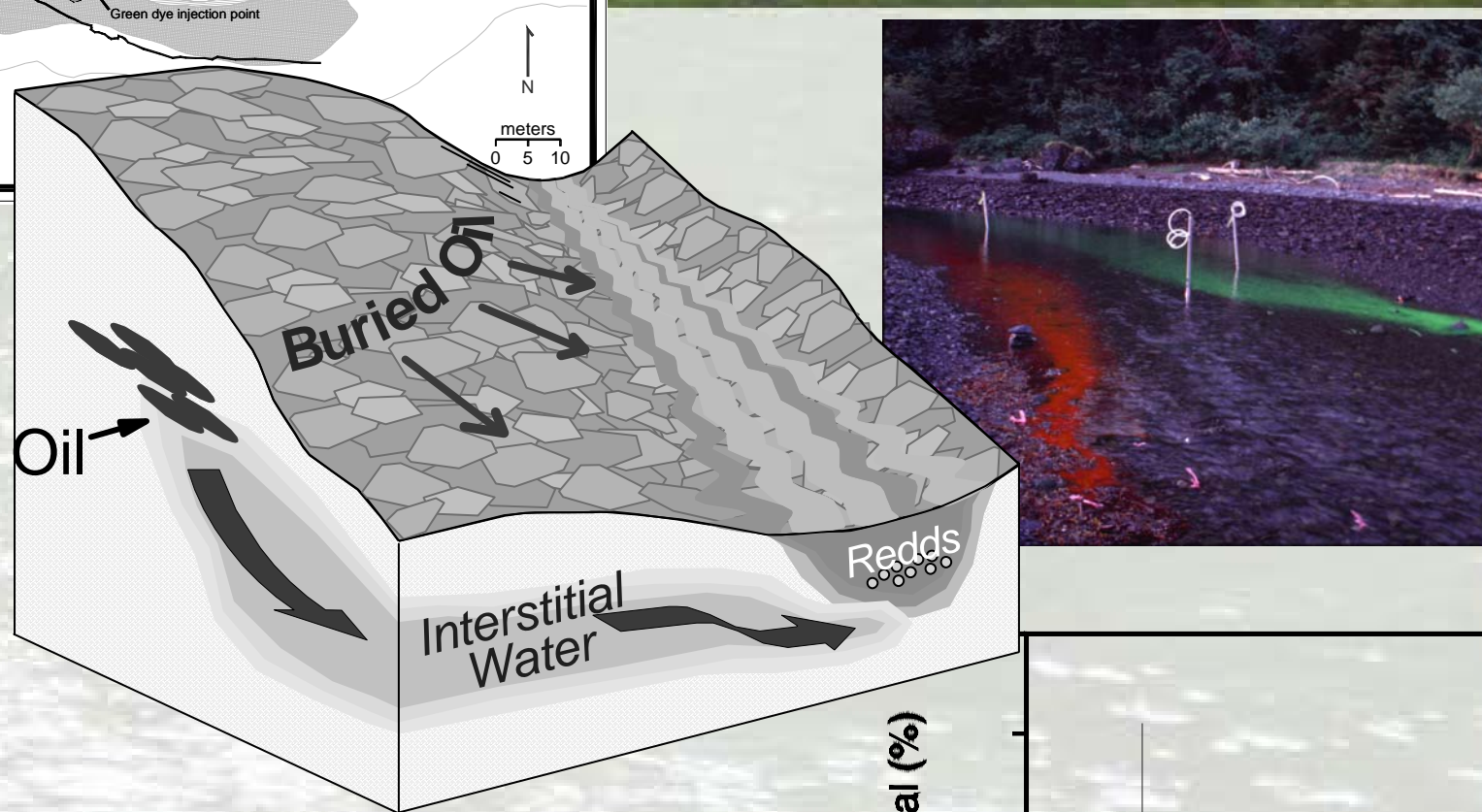
**(5) Evidence of exposure.** Induction of cytochrome P4501A (CYP1A) in pre-emergent pink salmon was evidence of exposure to PAH in Prince William Sound. Induction extended to at least 1991, the last year of study (Wiedmer et al. 1996).



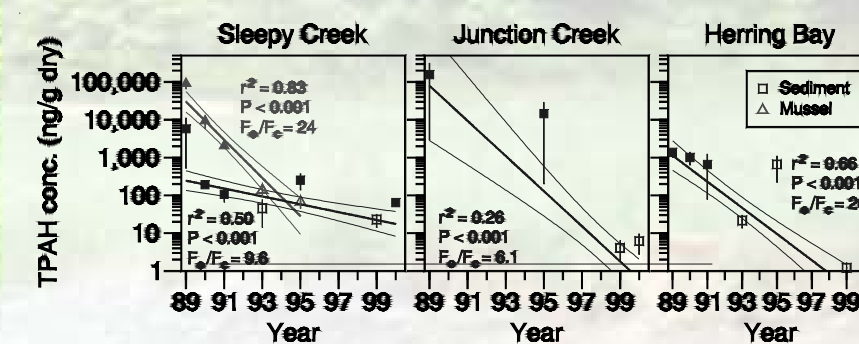
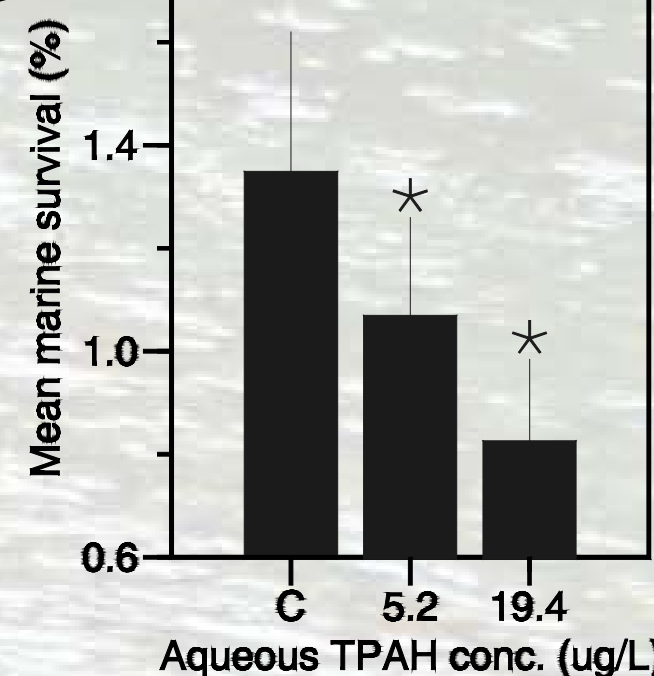
**(6) Evidence of effect (Prince William Sound).**

Embryo mortality was constantly greater in oiled streams than in reference streams through 1993 (Bue et al. 1996, 1998) Egg mortality was consistent with drainage of oil-contaminated water from surrounding sediment and oil was identified as causal. (Rice et al. 2001, Carls et al. 2003).

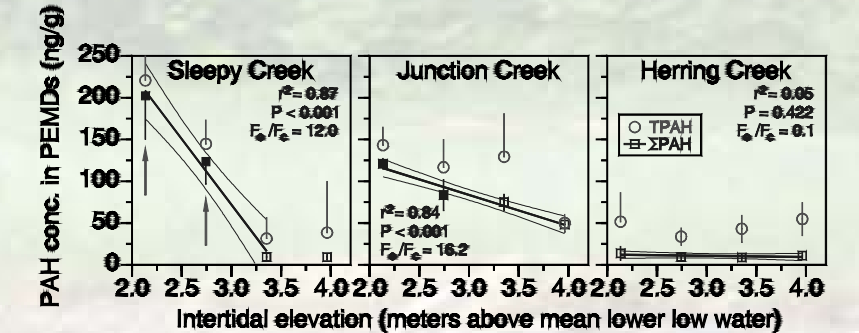
**(3) Exposure mechanism.** Oil-contaminated water drained from beaches into streams, exposing developing pink salmon embryos to toxic polynuclear aromatic hydrocarbons (PAH). Using red (rhodamine) and green (fluorescein) tracer dyes, Carls et al. (2003) confirmed drainage of interstitial water from surrounding beaches into substream water where pink salmon eggs incubate.



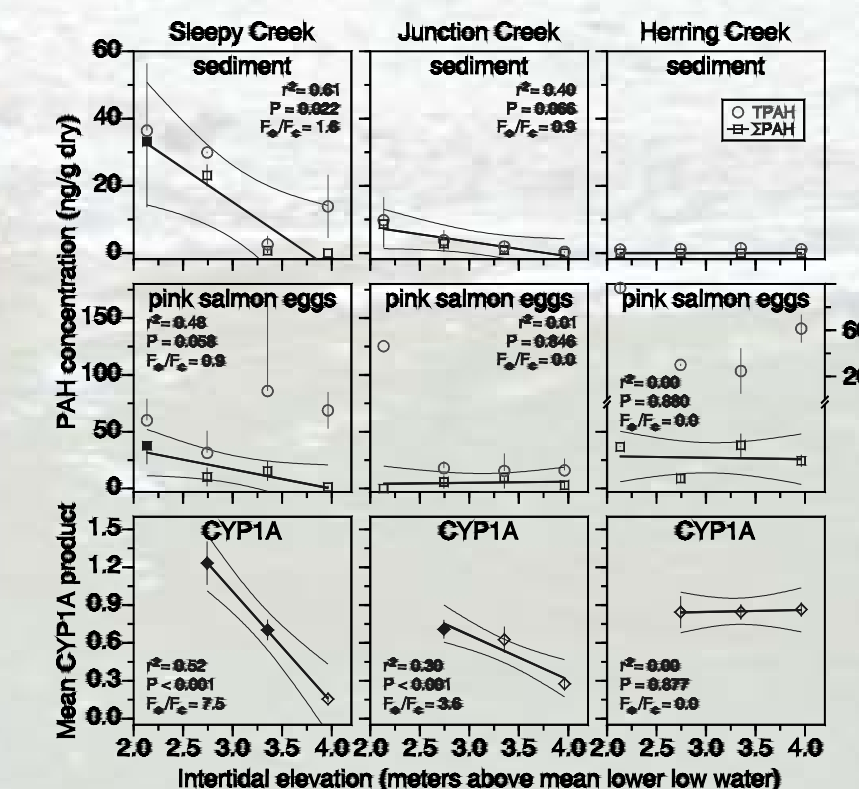
**(4) Evidence of effect (laboratory).** PAH dissolved from oil-coated sediment into water is toxic to pink salmon embryos at part-per-billion (ug/L) concentrations. (Marty et al. 1997; Heintz et al. 1999, 2000). The consequences of experimental embryonic exposure were evident throughout the life cycle and marine survival was reduced (Heintz et al. 2000).



**(7) Evidence of recovery.** Oil concentrations in intertidal sediment surrounding streams declined with time. Concentrations in 1995 were characterized by Murphy et al. (1999) as below the level expected to be toxic to pink salmon embryos. PAH concentration declines in sediment surrounding streams were consistent with declines in mussel beds and PAH became progressively less biologically available to mussels (Carls et al. 2001). However, known deposits of oil remain in intertidal sediment (Short et al. 2002), including deposits observed within 52 m of Sleepy Creek in 1999 (Carls et al. 2003).



**(8) Lingering oil was detected in some previously oiled streams a decade after the spill.** In 1999, PAH were significantly elevated in two of six previously heavily oiled streams in Prince William Sound in a pattern consistent with downstream drainage of oil-contaminated water. *Exxon Valdez* oil was identified in one of these streams (arrows; Carls et al. in press a). TPAH = total polynuclear aromatic hydrocarbons; sigmaPAH = TPAH minus naphthalene and C1-naphthalene. These hydrocarbons were sampled with low-density polyethylene passive sample devices (Carls et al. in press b).



**(9) PAH were bioavailable in some streams a decade after the spill.** In 1999, PAH levels in sediment and CYP1A induction in pink salmon embryos were correlated with PAH concentrations in stream water. PAH levels in tissue were not correlated, probably because of metabolism (Carls et al. in press a).

**(10) Conclusion: pink salmon habitat is recovering.**

Because 1999-2000 stream samples were all selected from previously heavily oiled streams, because oil was not detected in four of these streams, and because hydrocarbon concentrations surrounding all these streams declined exponentially in the decade after the *Exxon Valdez* oil spill, we infer that most pink salmon spawning habitat either has recovered or is recovering.